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EXAMINER	
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ART UNIT	PAPER NUMBER
1633	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/560,250	NEMEROW ET AL.	
	Examiner	Art Unit	
	FEREYDOUN G. SAJJADI	1633	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *29 May 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-7,10-54,57-68 and 72-85* is/are pending in the application.

4a) Of the above claim(s) *1-7,10-54,57,59-68 and 72-79* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *58 and 80-85* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Claim Status

Applicants' amendment dated May 29, 2009 that includes a response to the non-final action dated January 29, 2009, has been entered. No claims have been amended or cancelled. Claims 83-85 were newly added. Accordingly, claims 1-7, 10-54 and 57-68 and 72-85 are pending in the application. Claims 1-7, 10-54, 57, 59-68 and 72-79 stand withdrawn from further consideration with traverse. Applicants should note that the instant claims have been examined commensurate with the scope of the elected invention and the species of the invention, i.e. the last repeat of Ad37 (serotype D) and its modified form presented as SEQ ID NO: 48.

Claims 58 and 80-85 are under current examination.

Response & Maintained Claim Rejections - 35 USC § 103

Claim 58 stands rejected under 35 U.S.C. §103(a) as being unpatentable over Vigne et al. (U.S. Patent No.: 6,455,314; effective filing date: Aug. 27, 1998), in view of Hallenbeck et al. (U.S. Patent No: 2002/0137213; effective filing date June 2, 2000). The rejection set forth on page 6 of the Office action dated June 11, 2008, and pp. 3-4 of the previous Office action dated January 29, 2009 is maintained for reasons of record.

The Rejection:

The instant claims encompass an adenovirus particle comprising a fiber shaft protein modified in a last full repeat and a further modification in the fiber knob that include KO1.

Vigne et al. describe targeted adenovirus vectors for delivery of heterologous genes, wherein modifications of the internal sites of the adenovirus fiber protein that include short targeting peptides fused to the C-terminus of the fiber protein, or the fiber HI loop (knob) target the modified adenoparticles to specific cell types (Title and Abstract). Specifically disclosing that the fiber protein can be modified to have a fiber shaft that is shorter than a wild-type fiber

shaft, in particular by an in-frame deletion or by replacing it with the shaft from another serotype (column 6). In Example 3, Vigne et al. teach a shortened Ad5 shaft that retained only 6 or 9 repeats instead of 22 in the native protein (column 30), and additionally teach using SOE35Kg primer corresponding to the last repeat of the Ad3 fiber shaft and primers that include modifications resulting in the creation of restriction sites to generate an intertypic fiber composed of the Ad5 tail, the Ad3 shaft and part of the Ad5 knob, and flanked with unique restriction sites (columns 31 and 32, bridging). The disclosed mutation thus encompasses a substitution or replacement of the Ad5 shaft with Ad3, comprising a modification in the last full repeat of the fiber shaft. One mutant adenovirus thus generated (vBS1) was noted to bind less efficiently to CAR (column 33). Vigne et al additionally teach that at least a part of the fiber HI loop (knob) is replaced with a ligand peptide or targeting sequence, so as to functionally display its binding specificity at the capsid surface, that may comprise deletion of about 6 to 17 amino acids from the hexon HI loop, preferably not exceeding 11 amino acids (column 4). Further teaching: "Capsid modifications that impair the native entry pathway (e.g. fibers displaying short shafts) can therefore be combined with capsid modifications that provide an additional, CAR-independent, pathway of infection." (columns 47 and 48; bridging).

Vigne et al do not specifically describe the KO1 fiber knob mutation. Hallenbeck et al. describe adenovirus particles mutated in their fiber proteins that no longer bind to their natural cellular receptor and can be retargeted to a specific cell type through the addition of a ligand to the virus capsid (Abstract). Hallenbeck et al. specifically described are adenoviral constructs containing the KO1 fiber AB loop mutation (Fig. 9), displaying a diminished interaction with CAR (paragraph [0092])), thus providing for the deficiency of KO1 modification in the teachings of Vigne et al., and additionally providing the motivation to introduce the KO1 modification in the fiber knob region. Adenoviral vectors containing the KO1 mutation in conjunction with a ligand targeting moiety are described in Example 3.

Therefore, it would have been *prima facie* obvious for a person of ordinary skill in the art, to combine the teachings of Vigne et al. and Hallenbeck et al. to introduce the KO1 mutation as the fiber knob mutation in a retargeted adenoviral vector, as instantly claimed, with a reasonable expectation of success, at the time of the instant invention. A person of ordinary skill

in the art would have been motivated to introduce the KO1 modification in the fiber knob as taught by Hallenbeck et al., because such mutations would provide an additional CAR-independent pathway of infection for adenovirus retargeting.

Response to Arguments:

Applicants traverse the rejection, arguing that Vigne does not teach or suggest a modified adenovirus fiber in which the modification is in the last full β repeat. Further arguing that Vigne describes large deletions or substitutions involving all or most of the Ad5 fiber shaft protein. For example, Vigne describes viruses having modified Ad5 fibers in which large portions of the fiber shaft (i. e., repeats 4-16 or repeats 4-19, termed viruses "vBS 1" and "vBS2", respectively) are deleted, but the native last full repeat is left intact. Alternatively, Vigne demonstrated a substitution of the entire Ad5 fiber shaft with the fiber shaft of Ad3 (termed "vBII").

Applicants' arguments have been fully considered, but are not found persuasive. Applicants have admitted on the record that Vigne describes large deletions or substitutions involving all or most of the Ad5 fiber shaft protein. Thus, a deletion or substitution of all fiber shaft protein necessarily includes the last full β repeat. Moreover, Vigne et al. specifically state: "The method for targeting a specific cell type in accordance with the invention can be further enhanced by shortening the fiber protein shaft, e.g. such that the fiber shaft only contains repeats 1 to 3 and 17 to 22 of Ad5; repeats 1to 3 and 20 to 22 of Ad5; or with an Ad3 shaft." (column 7, lines 35-39). In Example 3, Vigne et al. teach a shortened Ad5 shaft that retained only 6 or 9 repeats instead of 22 in the native protein (column 30), and additionally teach using SOE35Kg primer corresponding to the last repeat of the Ad3 fiber shaft and primers that include modifications resulting in the creation of restriction sites to generate an intertypic fiber composed of the Ad5 tail, the Ad3 shaft and part of the Ad5 knob, and flanked with unique restriction sites (columns 31 and 32, bridging). The disclosed mutation thus encompasses a substitution or replacement of the Ad5 shaft with Ad3, comprising a modification in the last full repeat of the fiber shaft.

Applicants argue that the references provide no motivation to combine them nor would the skilled artisan have had a reasonable expectation of success in achieving an adenovirus

particle of the present claims because Vigne teaches away from adenovirus particles having the recited fiber shaft modification.

In response it should be noted that the teachings of Vigne et al. specifically include replacement of the entire shaft region and thus do not constitute any teaching away, contrary to Applicants' assertion. Further, the teachings of Vigne et al. and Hallenbeck et al. are both directed to introducing mutations in the adenovirus fiber proteins that alter CAR receptor binding. Thus, it would have been *prima facie* obvious for a person of ordinary skill in the art, to combine their respective teachings to introduce the fiber knob mutations in a retargeted adenoviral vector, as instantly claimed, with a reasonable expectation of success, at the time of the instant invention. A person of ordinary skill in the art would have been motivated to introduce modifications in both the fiber shaft and fiber knob because such mutations would likely be additive in affecting CAR receptor binding. Indeed, Vigne et al. describe combining mutations in the fiber shaft with capsid modifications to provide additional CAR-independent pathways for infection (columns 47 and 48 bridging), and further describe deletion of the fiber HI loop (knob protein; Example 2, column 26). Vigne el al. further describe replacement of a part of the fiber 1-11 loop (knob) with a ligand peptide or targeting sequence, that impair the native entry pathway and provide an additional, CAR-independent, pathway of infection." (columns 47 and 48; bridging). Hallenbeck et al. describe adenovirus particles mutated in their fiber proteins that no longer bind to their natural cellular receptor and can be retargeted to a specific cell type through the addition of a ligand to the virus capsid (Abstract). Hallenbeck et al. specifically describe adenoviral constructs containing the KOI fiber AB loop mutation (Fig. 9), displaying a diminished interaction with CAR (paragraph [0092]). Adenoviral vectors containing the KOI mutation in conjunction with a ligand targeting moiety are described in Example 3.

Thus, the rejection of claim 58 is maintained for reasons of record and the preceding commentary.

Response, Maintained & New Claim Rejections - 35 USC § 103

Applicants' claim amendments have necessitated the following new grounds of rejection.

Claims 58 and 80-82 stand rejected and claims 84 is newly under 35 U.S.C. §103(a) as being unpatentable over Vigne et al. (U.S. Patent No.: 6,455,314; effective filing date: Aug. 27, 1998), in view of Hallenbeck et al. (U.S. Patent No: 2002/0137213; effective filing date June 2, 2000), as applied to claim 58 above, and further in view of Havenga et al. (U.S. Patent Publication No: 2003/0017138; filed Jul. 7, 1999). The rejection set forth on pp. 5-7 of the previous Office action dated January 29, 2009 is maintained for claims 58 and 80-82, and is further applied to new claim 84 for reasons of record.

The claims encompass an adenovirus particle comprising a modified adenovirus fiber in which modification is a mutation, insertion or replacement of at least one amino acid in a fiber shaft β-repeat corresponding to the last full β-repeat, and wherein the fiber further comprises a modification in the AB loop or the CD loop of the fiber knob, wherein the fiber knob modification is selected from the group consisting of K01 and K012, whereby binding of the modified fiber to CAR is reduced, and wherein the modification comprises replacement of the last full β-repeat with a corresponding repeat sequence form an Ad37 serotype D adenovirus, as set forth in SEQ ID NO: 48.

Vigne et al. describe targeted adenovirus vectors for delivery of heterologous genes, wherein modifications of the internal sites of the adenovirus fiber protein that include short targeting peptides fused to the C-terminus of the fiber protein, or the fiber HI loop (knob) target the modified adenoparticles to specific cell types (Title and Abstract). Specifically disclosing that the fiber protein can be modified to have a fiber shaft that is shorter than a wild-type fiber shaft, in particular by an in-frame deletion or by replacing it with the shaft from another serotype (column 6). Additionally disclosing substitution or replacement of the Ad5 shaft with Ad3, comprising a modification in the last full repeat of the fiber shaft (column 33). Vigne el al. further describe replacement of a part of the fiber 1-11 loop (knob) with a ligand peptide or targeting sequence, that impair the native entry pathway and provide an additional, CAR-independent, pathway of infection." (columns 47 and 48; bridging). With respect to modification of the last full repeat, Vigne et al. teach the fiber shaft as comprising pseudorepeats of 15 amino acids, which are believed to form two alternating β–strands and β–bends; and that the overall length of the fiber shaft and the number of repeats varies between different adenoviral serotypes

(column 2, lines 22-30). Vigne et al. further teach that the fiber protein can be modified to have a fiber shaft that is shorter than a wild-type fiber shaft, in particular by an in-frame deletion or by replacing it with the shaft from another serotype (column 6). Additionally teaching using SOE35Kg primer corresponding to the last repeat of the Ad3 fiber shaft and primers that include modifications resulting in the creation of restriction sites to generate an intertypic fiber composed of the Ad5 tail, the Ad3 shaft and part of the Ad5 knob, and flanked with unique restriction sites (columns 31 and 32, bridging). The disclosed mutation thus encompasses a substitution or replacement of the Ad5 shaft with Ad3, comprising a modification in the last full repeat of the fiber shaft.

Hallenbeck et al. describe adenovirus particles mutated in their fiber proteins that no longer bind to their natural cellular receptor and can be retargeted to a specific cell type through the addition of a ligand to the virus capsid (Abstract). Hallenbeck et al. specifically describe adenoviral constructs containing the KOI fiber AB loop mutation (Fig. 9), displaying a diminished interaction with CAR (paragraph [0092]). Adenoviral vectors containing the KOI mutation in conjunction with a ligand targeting moiety are described in Example 3.

While Vigne et al. and Hallenbeck et al. do not specifically describe the serotype D Ad37 virus having the sequence set forth SEQ ID NO: 48, such adenovirus serotype and sequences of the last full repeat of fiber shaft were known in the prior art.

Havenga et al. describe chimeric adenoviruses as vectors, wherein the hybrid adenoviruses contain a genome derived from different adenovirus serotypes, displaying a modified host range that overcome the limitations with currently used serotype C adenoviruses (Abstract). Havenga et al. state: "Preferably, the (chimeric) adenoviruses capable of transducing a CAR negative cell include at least an adenovirus receptor binding part of a fiber protein from an adenovirus of subgroup D" (paragraph [0020], further depicting the fiber shaft sequences of type 37 in Fig. 7, and specifically describing Sequence 31, comprising the last full repeat of instantly claimed SEQ ID NO: 48.

Therefore, it would have been *prima facie* obvious for a person of ordinary skill in the art, to combine the teachings of Vigne et al., Hallenbeck et al. and Havenga et al. to substitute or modify the last full repeat of the fiber shaft of a serotype 37 in a retargeted adenoviral vector, as

instantly claimed, with a reasonable expectation of success, at the time of the instant invention. A person of ordinary skill in the art would have been motivated to introduce a modification in the fiber shaft as taught by both Vigne et al. and Havenga et al., because such mutations would provide an additional CAR-independent pathway of infection for adenovirus retargeting.

Response to Arguments:

Applicants traverse the rejection, arguing that Vigne does not teach or suggest the fiber shaft modification corresponding to the last full β-repeat. Rather, Vigne teaches large deletions or substitutions involving all or most of the Ad5 fiber shaft protein which may additionally comprise all or a portion of a last full 13 repeat but doest not target the specific last full β-repeat portion of the fiber to modify. Because of this, Vigne actually teaches away from focusing on modifying the last full β-repeat.

Applicants' arguments have been fully considered, but are not found persuasive. As previously indicated, language of instant claim 58 is nowhere limiting with respect to the size of the mutation, insertion or replacement, as "at least one amino acid" fails to set an upper limit on mutation size. With respect to modification of the last full repeat, Vigne et al. teach the fiber shaft as comprising pseudorepeats of 15 amino acids, which are believed to form two alternating β–strands and β –bends; and that the overall length of the fiber shaft and the number of repeats varies between different adenoviral serotypes (column 2, lines 22-30). Vigne et al. further teach that the fiber protein can be modified to have a fiber shaft that is shorter than a wild-type fiber shaft, in particular by an in-frame deletion or by replacing it with the shaft from another serotype (column 6). Additionally teaching using SOE35Kg primer corresponding to the last repeat of the Ad3 fiber shaft and primers that include modifications resulting in the creation of restriction sites to generate an intertypic fiber composed of the Ad5 tail, the Ad3 shaft and part of the Ad5 knob, and flanked with unique restriction sites (columns 31 and 32, bridging). The disclosed mutation thus encompasses a substitution or replacement of the Ad5 shaft with Ad3, comprising a modification in the last full β-repeat of the fiber shaft. Modifications of the fiber shaft protein that include the last full β-repeat thus cannot be considered a teaching away from modifying the

last full β-repeat. Therefore, Vigne et al teach all the limitation of the instant claims, contrary to Applicants' assertion.

Moreover, Havenga et al. depict the fiber shaft sequences of Ad 37 in Fig. 7, and specifically describe Sequence 31, comprising the last full β-repeat of instantly claimed SEQ ID NO: 48. Therefore any sequence modifications that include the last full β-repeat of the fiber shaft meet the limitation of claim 58.

Thus, the rejection is maintained for claims 58 and 80-82, and is further applied to new claim 84 for reasons of record and the foregoing remarks.

New Claim Rejections - 35 USC § 103

Applicants' claim amendments have necessitated the following new grounds of rejection.

Claims 58, 83 and 85 are newly under 35 U.S.C. §103(a) as being unpatentable over Vigne et al. (U.S. Patent No.: 6,455,314; effective filing date: Aug. 27, 1998), in view of Kaleko et al. (U.S. Patent Publication No: 2004/0002060; effective filing date Jan. 24, 2002) .

The claims encompass an adenovirus particle comprising a modified adenovirus fiber in which modification is a mutation, insertion or replacement of at least one amino acid in a fiber shaft β-repeat corresponding to the last full β-repeat, and wherein the fiber further comprises a modification in the fiber knob, that is K012.

Vigne et al. describe targeted adenovirus vectors for delivery of heterologous genes, wherein modifications of the internal sites of the adenovirus fiber protein that include short targeting peptides fused to the C-terminus of the fiber protein, or the fiber HI loop (knob) target the modified adenoparticles to specific cell types (Title and Abstract). Specifically disclosing that the fiber protein can be modified to have a fiber shaft that is shorter than a wild-type fiber shaft, in particular by an in-frame deletion or by replacing it with the shaft from another serotype (column 6). Additionally disclosing substitution or replacement of the Ad5 shaft with Ad3, comprising a modification in the last full repeat of the fiber shaft (column 33). Vigne el al. further describe replacement of a part of the fiber 1-11 loop (knob) with a ligand peptide or targeting sequence, that impair the native entry pathway and provide an additional, CAR-

independent, pathway of infection." (columns 47 and 48; bridging). With respect to modification of the last full repeat, Vigne et al. teach the fiber shaft as comprising pseudorepeats of 15 amino acids, which are believed to form two alternating β–strands and β–bends; and that the overall length of the fiber shaft and the number of repeats varies between different adenoviral serotypes (column 2, lines 22-30). Vigne et al. further teach that the fiber protein can be modified to have a fiber shaft that is shorter than a wild-type fiber shaft, in particular by an in-frame deletion or by replacing it with the shaft from another serotype (column 6). In Example 3, Vigne et al. teach a shortened Ad5 shaft that retained only 6 or 9 repeats instead of 22 in the native protein (column 30), and additionally teach using SOE35Kg primer corresponding to the last repeat of the Ad3 fiber shaft and primers that include modifications resulting in the creation of restriction sites to generate an intertypic fiber composed of the Ad5 tail, the Ad3 shaft and part of the Ad5 knob, and flanked with unique restriction sites (columns 31 and 32, bridging). The disclosed mutation thus encompasses a substitution or replacement of the Ad5 shaft with Ad3, comprising a modification in the last full repeat of the fiber shaft. One mutant adenovirus thus generated (vBS1) was noted to bind less efficiently to CAR (column 33). Vigne et al additionally teach that at least a part of the fiber HI loop (knob) is replaced with a ligand peptide or targeting sequence, so as to functionally display its binding specificity at the capsid surface, that may comprise deletion of about 6 to 17 amino acids from the hexon HI loop, preferably not exceeding 11 amino acids (column 4). Further teaching: "Capsid modifications that impair the native entry pathway (e.g. fibers displaying short shafts) can therefore be combined with capsid modifications that provide an additional, CAR-independent, pathway of infection." (columns 47 and 48; bridging).

Vigne et al do not specifically describe the KO12 fiber knob mutation. Kaleko et al. describe fiber shaft modifications for efficient targeting of adenoviral vectors, that can be combined with other modifications such as fiber knob modifications to produce fully ablated detargeted adenoviral vectors (Abstract). Kaleko et al. specifically describe the KO12 modification in paragraph [0040], curing the deficiency in the teachings of Vigne et al., and additionally providing the motivation to introduce the KO12 modification in the fiber knob region.

The teachings of Vigne et al. and Kaleko et al. are directed to combining both fiber shaft and fiber knob modifications in adenoviral vectors. Therefore, it would have been *prima facie* obvious for a person of ordinary skill in the art, to combine the teachings of Vigne et al. and Kaleko et al. to introduce the KO12 mutation as the fiber knob mutation in a retargeted adenoviral vector, as instantly claimed, with a reasonable expectation of success, at the time of the instant invention. A person of ordinary skill in the art would have been motivated to introduce the KO12 modification in the fiber knob, because such mutations would provide an additional CAR-independent pathway of infection for adenovirus retargeting, and is specifically taught by Kaleko et al.

Conclusion

Claims 58 and 80-85 are not allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. The claims are drawn to the same invention claimed earlier in the application and would have been finally rejected on the grounds and art of record in the next Office Action if they had been entered earlier in the application. Accordingly, **THIS ACTION IS MADE FINAL.** See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR§1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to FEREYDOUN G. SAJJADI whose telephone number is (571)272-3311. The examiner can normally be reached on 6:30 AM-3:30 PM EST.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Joseph Woitach can be reached on (571) 272-0739. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Fereydoun G Sajjadi/
Primary Examiner, Art Unit 1633